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Race, ethnicity, and sepsis: beyond adjusted odds ratios

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Critical Care Medicine has published at least 40 articles related to race and ethnicity since 1995. These are challenging studies, primarily from populations in the United States, that try to understand a well-documented problem: health outcomes and therapy vary considerably by race and ethnicity (1). Because race and ethnicity data collection are mandated under many programs, these studies are often easier to do, than to interpret. Although collection is mandated, accuracy and reliability is poor and may be particularly so in patients who die rapidly after an acute illness and are assigned a race by an observer (2). Even the terms “race” and “ethnicity” are subject to considerable debate (1). An increasing number of people define themselves as mixed race where only one may be allowed in data entry forms. Race and ethnicity are multidimensional traits, capturing nearly all of the determinants of health from gene to society. Race represents an exposure for external health risks posed by biological (genetic), environmental, social, and behavioral factors. Ethnicity is an artificial grouping, determined by shared culture, language, and/or national origin; overlaps with race and is a socio-political construct (2).

When studying race/ethnicity as exposures, it is important to understand the reasons for and methods used to collect these variables (2). For example, ethnicity is often collected as self-reported variable, which has limited concordance to administratively coded race/ethnicity designations (2). Thus, when race/ethnicity are studied as explanatory variables for population health disparities, the resulting associations and choice of confounders in these studies is not just about *causality* but about understanding the *causal pathway*. For example, if the association between race/ethnicity and sepsis outcomes disappear when adjusted for severity of illness, but higher severity of illness on admission is seen in blacks who are at

greater risk of becoming septic, which could be interpreted in different ways, depending on how the research question was framed. Similarly, these associations between race/ethnicity potentially reflects differences in access to health care, quality of health care received, exposure to occupational or environmental hazards, other unknown factors and combinations thereof (2). However, the access to health care and occupational or environmental risk overlaps with socio-economic status, which in turn is associated with educational attainment that is related to occupation and health-state. Therefore, studying how health outcomes and therapy vary considerably by race and ethnicity are challenging research questions, with often tacit underlying assumptions.

Race/ethnicity may be a risk factor for sepsis, as the incidence or prevalence of infection or sepsis is higher in one racial/ ethnic group than in other groups(3), but the likelihood of developing sepsis varies considerably among members of the same racial/ ethnic group. Importantly, the racial/ethnic group studied as a population at greater risk of sepsis and related outcomes share many process of care characteristics with people in other racial/ethnic groups (2). For example, beginning with observations by Kahn and colleagues(4), studies have demonstrated that differences in process of care or outcome by race were attenuated after accounting for the hospital or clinician the patient saw. For example, while overall blacks seemed to get more intensive care than whites at the end-of-life, this was more a function of where they received their care than that they were black. So, while blacks were more likely to receive their care at hospitals that provided more intensive end-of-life care, whites also received more intensive care at these hospitals (5). It is also well recognized that discharge coding for sepsis differs between hospitals and an argument that has been considered in this context is the potential differences

sepsis coding by race or ethnicity (6). To be clear, this is still a form of racial disparity, but it lends itself to an entirely different set of explanations and solutions than disparate care at the individual patient level.

In this issue of CCM, Chaudhary NS et al, aimed to determine the racial disparities in severe sepsis hospitalizations and outcomes. They restricted the study population to academic medical centre affiliated hospitals in the Vizient Consortium. The authors highlight access to a more recent and nationally representative cohort as their key motivation and strength of this work. Sepsis hospitalizations and adjusted odds of hospital mortality were lower in patients coded as Black, which is contrary to reports where standardized population-based incidence rates were used (3). The authors' decision to separate infection by community acquired, healthcare-associated, and hospital-acquired was sensible because they did not exclude sepsis rehospitalisation from the dataset and as race /ethnicity is associated with nosocomial infections.

Rather than repeatedly highlighting race /ethnicity healthcare disparities in sepsis (3, 7, 8), we propose that ICU researchers could use theoretic models (9) to explain the mechanisms underpinning these health disparities, as these disparities are not unique to sepsis. Despite years of research, variations in patterns of human genome DNA sequences between race does not explain the health disparities in sepsis, as the gene pools and polymorphisms do not change much in decades, whereas socioeconomic status and health behaviors often do (9, 10). Socioeconomic status and health behaviors could explain race/ethnic health disparities in sepsis, as they are associated with other risk factors for sepsis such as alcohol intake, smoking, comorbidity, access to health care and process of healthcare delivery. The Institute of Medicine report from 2003 clearly highlights the impact of race/ethnicity

on the structure and process of healthcare delivery (1). Exploring theoretical models(9) with quantitative methods requires prospective hypothesis driven data collection; as retrospective administrative dataset studies, have limited information on clinical-decision making processes and their relationship to standards of care provided. Equally, such studies, could explore, how patients themselves influence the decisions made by the healthcare provider, which will highlight the true directionality of these *causal pathways*.

In summary, adjusted odds ratios for mortality and incident rates will not help us change health disparities. By inadvertently focusing on broad race/ethnicity grouping within administrative datasets in retrospective studies, we risk inadequately recognizing the underlying *causal pathways*, that could inform public health policy to address these health disparities.

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